--25. A method for inducing the production of CalbindinD-28K wherein said method comprises administering to a patient a composition according to claim 22.--

--26. A method for inducing the production of CalbindinD-28K wherein said method comprises administering to a patient a composition according to claim 23.--

--27. A method for inducing the production of CalbindinD-28K wherein said method comprises administering to a patient a composition according to claim 24.--

--28. A method for improving or treating cerebral function and/or organic function disorders wherein said method comprises administering to a patient a composition according to claim 22.--

--29. The method of claim 28, wherein said cerebral function disorders are caused by ischemic disorders.--

--30. The method of claim 29, wherein said ischemic disorders are selected from the group consisting of cerebral infarction, intracerebral hemorrhage and cerebral arteriosclerosis.--

--31. The method of claim 28, wherein said organic function disorders are selcted from the group consisting of senile dementia, cerebral injury, cerebral operation, Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis.--

--32. A method for improving or treating cerebral function and/or organic function disorders wherein said method comprises administering to a patient a composition according to claim 23.--

A2 --33. The method of claim 32, wherein said cerebral function disorders are caused by ischemic disorders.--

--34. The method of claim 33, wherein said ischemic disorders are selected from the group consisting of cerebral infarction, intracerebral hemorrhage and cerebral arteriosclerosis.--

--35. The method of claim 32, wherein said organic function disorders are selcted from the group consisting of senile dementia, cerebral injury, cerebral operation, Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis.--

--36. A method for improving or treating cerebral function and/or organic function disorders wherein said method comprises administering to a patient a composition according to claim 24.--

--37. The method of claim 36, wherein said cerebral function disorders are caused by ischemic disorders.--

--38. The method of claim 37, wherein said ischemic disorders are selected from the group consisting of cerebral infarction, intracerebral hemorrhage and cerebral arteriosclerosis.--

--39. The method of claim 36, wherein said organic function disorders are selcted from the group consisting of senile dementia, cerebral injury, cerebral operation, Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis.--

--40. A method for selecting a neuroprotective compound, wherein said method comprises evaluating the activation of receptors for various kinds of physiologically active substances and the phosphorylation of the FGF receptor caused by the induction of CalbindinD-28k production.--

--41. The method for selecting a neuroprotective compound according to claim 40, wherein said method comprises evaluating the autophosphorylation of the FGF receptor.--

--42. The method for selecting a neuroprotective compound according to claim 40, wherein said method is performed by evaluating for neuroprotective effect of the

physiologically active substance against glutamate-induced neurodegeneration together with one or more of the following tests (i)-(iii):

(i) evaluating for antagonism against the neuroprotective effect of the physiologically active substance by treatment with MTA (5-deoxy-5-methyl-thioadenosine), which inhibits autophosphorylation of the FGF receptor, and by treatment with inhibitors of various physiologically active substance receptors, to determine if the neuroprotective effect is due to autophosphorylation of receptors of the FGF receptor;

(ii) evaluating the CalbindinD-28k inducing effect of the physiologically active substance; or

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(iv) confirming that the neuroprotective effect of the physiologically active substance is due to its inducing CalbindinD-28k production, by treating with the antisense oligonucleotide of CalbindinD-28k and determining if CalbindinD-28k production is antagonized.--

--43. The method according to claim 18, wherein said physiological active substance receptors are selected from the group consisting of receptors for neurotrophin-3 (NT-3), neurotrophin-4/5 (NT-4/5), brain-derived neurotrophic factor (BDNF), insulin-like growth factor-I/II (IGF-I/II), platelet-derived growth factor (PDGF), and estrogen.--

--44. A neuroprotective compound selected by the method according to claim 40.--

--45. A composition comprising a neuroprotective compound according to claim 44.--

--46. A method of treating or improving cerebral functional disorders and/or cerebral organic disorders, wherein said method comprises administering the composition according to claim 45 to a patient in need thereof.--

--47. The method according to claim 46, wherein said cerebral functional disorders are caused by ischemic disorders.--

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--48. The method according to claim 47, wherein said ischemic disorders are selected from the group consisting of cerebral infarction, intracerebral hemorrhage and cerebral arteriosclerosis.--

--49. The method according to claim 46, wherein said cerebral organic disorders are selected from the group consisting of senile dementia, cerebral injury, cerebral operation, Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis.--